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TITLE:

COMBINED EFFECTS OF BALLISTICS INJURY AND BLOOD LOSS

PRINCIPAL INVESTIGATOR: 1.C.P. SENGCHSKI

PI ADDRESS:

UNITE DE CHIRURGIE ET DE PHYSIOLOGIE EXPERIMENTALE

INSTITUT DE MEDECINE TROPICALE DU SERVICE DE SANTE DES AMBEES

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COMBINED EFFECTS OF BALLISTICS INJURY AND BLOOD LOSS

I.C.P. SENBOWSKI (1)
Ph. D. P. E. MARTIN (2)
J.I. MUTKLLEC (3)
J.P.I. BRETTAU (4)

- (1) Vétérmaire Biologiste Principal. Unité de Chirurgie et de Physiologis expérimentale. Centre d'Enidez et de Recherche en Médecine Tropicale. Institut de Médecine Tropicale du Service de Santé des Armées -MARSEILLE - FRANCE
- (2) Vétérmaire Biologiste. Unité de Chirurgie et de Physiologie expérimentale. Contre d'Études et du Rechtsche en Médecine Tropicale. Institut de Médecine Tropicale du Service de Semé des Armétes MARSERLE FRANCE.
- (3) Pharmacien Chimiste en Chaf. Unité de Chimisgie et de Physiologie expérimentale, Coutre d'Étades et de Recherche en Médacine Tropicale. Institut de Marie Tropicale du Service de Serné des Armées MARSEILLE FRANCE
- (4) Vétérinaire Biologiste. Chef des Services. CERML Université d'AIX en PROVENCE, MARSEILLE FRANCE

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classic hemorrhage model does not therefore exactly represent the hemorrhage received in combat which is generally associated with a ballistic injury.

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COMBINED EFFECTS OF BALLISTICS INJURY AND RLOOD LOSS

I- INTRODUCTION

Recent studies suggest that a variety of factors may impact or the physiological and biochemical responses to and survival following a major hemorrhage. For example, in response to removal of a fixed volume of blood, animals bled in a holding cage had a higher survival rate than those bled in a Pavlov sling (6). This simple change in handling procedure drastically changed the outcome. In addition, it has been found that the hemodynamic and biochemical responses during recovery from blood loss are altered by the addition of sciatic nerve stimulation as a nociceptive input, showing that possible nervous system involvement from injury can modify the homeostatic reflexes to hemorrhage (18). A high velocity shot in the thigh of an aneathetized pig induces a high frequency, high amplitude pressure wave. This wave is not restricted to the injury area, but can be recorded throughout the body and induces structural changes in the sciatic nerve and even in the central nervous system (22, 9).

These findings raise questions of the applicability of information obtained from pure hemorrhage snimal models to the understanding of what happens to the military trauma victim, who would likely have ballistic injury plus hemorrhage. Almoskog (1) studied the effects of hypovolemia on local missile injury, but he did not study the opposite question, how does local missil injury affect the responses to hypovolomia? Therefore, our study was designed to characterize the physiological responses involved in the combination of a realistic battlefield ballistic injury with a fixed-volume hemorrhage. This information should help us more fully understand the applicability of animal models to combat casualty care.

Hemorrhage model:

Previous studies demonstrated that the domestic pig is superior to the dog or sheep as a large animal model of hemorrhagic hypotension (23, 7, 12). Functional characteristics of the porcine cardiovascular system and the pig's hemodynamic responses to hemorrhage more closely resemble those of the human than do other animal models (14, 28). Moreover, an animal with sufficiently large-sized muscle mass is required in order to perform the the type of ballistic, soft tissue injury utilized in this study. The Large-white pig, weighing approximately 90 kg meets this muscle mass requirement.

It is necessary to splenectomize the pig, because the pig has a large contractile spleen which Hannon and al (11) showed is capable of storing a large volume of red blood cells which can be released in response to hemorrhage. The splenectomized pig has responses to hemorrhage that are remarkably similar to those reported for adult humans (13).

The fixed-volume hemorrhage model seems to simulate the hemorrhage seen in combat casualties better than the Wiggers fixed-pressure hemorrhage model (10). Also, recent experiments have demonstrated that blood loss produced by aortotomy results in a more hypotensive response than that expected by the loss of a similar volume of blood in the fixed-volume phlabotomy model (3). This is another indication that injury plus blood loss alters the responses in the combat casualty.

II- KXTHODG

Surgical preparation:

Thrirty-eight female Large-white pigs (obtained from a commercial swine farm) were used in this study. They underwent three operations. The animals were fasted 24 hours before each surgery with water available ad libidum. On the morning of surgery, they were pre-medicated with ketamine (30 mg/kg), acepromazine (2 mg/kg) and atropine (0,05 mg/kg). As intravenous infusion of saline (0,9 % NaCl) was begun and the animals were intubated. For major surgery, 0,5% - 1.0% halothene was administered with 20% 02 and 80% N2O, with a Minerve respirator. All surgery was performed using aseptic techniques.

The three surgeries were as follows: 1) Six weeks before the experiment, the spleen was removed via a left posterior lateral hypochondral laparotomy.

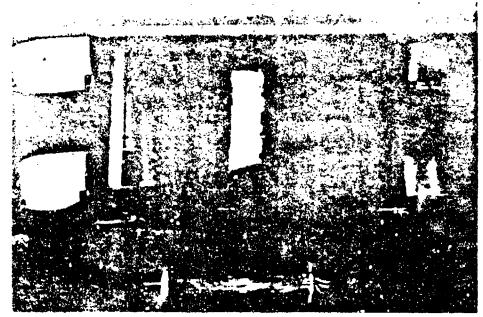
2) Two weeks before the experiment, we chronically implanted a renal flow probe, catheters and EKG electrodes. The electromagnetic flow probe (4 mm, Spectramed) was placed around the left renal artery via a retroperitoneal incision. Two silicon catheters (Silastic N°602 25; Dow Corning) were inserted non-occlusively; one was inserted into the left carotid artery to be used for taking blocd samples, and the other into the left external jugular vein and advanced to the right atrium to be used for measuring central venous pressure (Statham transducer - Spectramed). Three EKG leads (spiral of steel wire on silastic patch) were placed subcutanously in the middle of the right and left chest and on the back and attached to the musculature. 3) One week before the experiment, we performed a left thoracotomy and an electromagnetic blood flow transducer (20 mm -Spectramed) was placed around the aortic root. A pressure transducer (Roenigbberg Instrument) was also placed into the aortic root. A strain quuqe embedded in silastic (Madical Adhesiva Silicon - Dow Corning) was sutured onto the left ventricular myocardium. Finally, a largu-bore silastic catheter (nº 602 405, Dow Corning), through which the hemorrhage was performed, was sutured nonocclusively into the aortic wall.

The catheters and the flow probe, pressure transducer and EKG connectors were tunneled subcutaneously and exteriorized out the left side and sutured at an interscapular position. A specially designed protective box with connectors was placed on the pig's back: it enabled us to record from the unanesthetized animal (photo 1).

Catheters were flushed daily with a normal saline solution and filled with a saline solution containing heparin (10 U/ml). Exytetracyclin (50 mg/10 kg) was administered daily to prevent infection. The entire post-operative recovery was munitored and appropriate treatment administered if needed.

Experimental protocol:

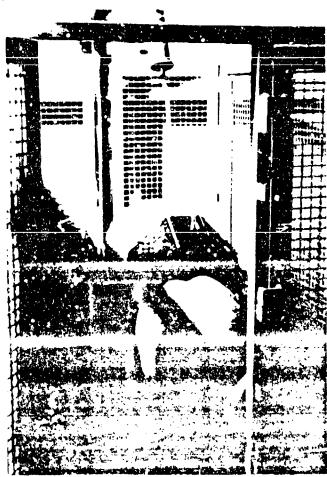
There were 5 experimental groups with 5 animals per group and the animals were randomly assigned to each group:



a- Protective box and special turning connector
The protective box have three partitions (on left: for
catheters, on right: for acquiring phyriological datas, on
middle: for hemorrhage catheter)
The special turning connector have 50 connections.

b- The physiologicals parameters are recorded on conscious animals thanks to a special turning connectors placed on the back of the pig.

PROTO nº1



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croup 1: anesthetized pigs (A)
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To achieve the ballistic injury, we used unfragmentable high velocity bullets (similating AK 74 round). These bullets were reconstituted according Fackler's technique (8). The target area for the shot was in the thigh muscle. The target area was determinated by radiography in order to avoid any fracture, large vessel or nerve damage. This standardized shot procedure also resulted in a standardized missile trauma (4).

We removed 15 ml/kg blood continuously over 60 min, in a logarithmic fashion to mimic a hemorrhage from a severed artery: the blood was removed uniformly in equal parts over succesive intervals of 9, 10, 12, 13 and 16 minutes (10, 11).

The pigs were fasted 12 h prior to experiment with water available ad lib. On the morning of the experiment, the pigs were brought to a holding cage to which they had previously been trained to become familiarized with the surroundings and used to the experimental procedure. The acrtic and renal flow transducers were connected to the blood flowmeter (2202 Spectramed). The acrtic pressure transducer and venus pressure transducer were connected to amplifiers (Signal Conditioning Amplifier 2310, Vishay Measurements). The EKG electrodes were connected to the amplifier (Universal Amplifier, Gould). All physiological parameters were recorded on a Dash IV recorder (Astro-Med).

A period of 30 min was allowed for the animal to become accustomed to the surroundings before performing the initial mesurements and taking any samples. After this 30 min period, baseline control measurements and a blood sample was taken (t-30). After the recording and samples were taken, the anesthetized groups received ketamine (10 mg/kg bolus iv and 0,5 mg/kg/min infusion). Then, they were rapidly taken to the ballistic laboratory. The post-anesthetic

group 2. anesthetized and injured pigs (AI)

group 3: anesthetized and bled pigs (AB)

group 4: anesthetized, injured and bled pigs (AIL)

group 5: conscious bled pigs.(CB)

measurements and sample were then obtained (t-15). From that moment, the physiological parameters were continuously recorded until the end of the hemorrhage. In the groups receiving the ballistic injury, it preceded the third sample (t-5) and the start of hemorrhage (t-0). Samples were taken at 5, 9, 19, 31, 44 and 60 min; and physiological recordings were taken at 120, 180 and 36, min after the beginning of the hemorrhage. The anesthetic influsion was stopped at 60 min. These recording and sampling procedures were the same for each group. After the injury was induced, local treatment consisting of antisepsis and sticking plaster was carried out. Samples and recordings were made daily for four days in order to study the recovery. After the experiment, the animals were euthanized with an intravenous overdose of barbiturate.

Sample analyses :

Each sample require 40 ml of blood. Hematology - re studied with a Coulter Counter T540. Coagulation tests were performed with a fibrometer (Merieux). Blood gases were mesured with an ABL 500 Blood Gas Analyzer (Radiometer). Biochemical analyzes were made with a semi-automatic analyzer FP 900 (Labsystem), and ionograms with an automatic analyzer M644 (Ciba-Corning). Finally, catecholamines were measured by high-pressure liquid chromatography (Varian 5-J LC-Chromatofield Eldec 103).

Physiological parameters :

Heart rate, cardiac output, renal blood flow, and arterial and venous pressure were directly recorded (see above). Systemic vascular resistance was calculated by dividing the mean arterial pressure by the cardiac output. Renal vascular resistance was estimated as the ratio of mean arterial pressure to renal blood flow. Stroke volume was calculated with the fraction: cardiac output/heart rate.

Statistical annlysis :

The homogeneity of the baseline values was tested with a Friedman pseudo two-way ANOVA (21,17). To ascertain whether there were changes within a group with time, we performed a Wilcoxon signed rank test comparing post-hemorrhage values to the control values.

limit, we determined whether there were differences between groups using the Mann-Whitney U test. In order to elucidate how ballistic injury modified the effects of hemorrhage, we made the following comparisons:

- 1) To determine the effect of hemorrhage alone in anesthetized animals, we compared the non-injured, non-bled group (A) with the non-injured, bled group (AB).
- 2) To determine the effect of injury alone in anesthetized animals, we compared the non-injured, non-bled group (A) with the injured, non-bled group (AI).
- 3) To determine the effect of the combined injury and hemorrhage in anesthetized animals, we compared the non-injured, non-bled group (A) with the injured, bled group (AIB).
- 4) To determine the effect of hemorrhage on the injured animal, we compared the injured. non-bled group (AI) with the injured, bled group (AIB).

Lestly, we analyzed the effects of anesthesia on the hemorrhage response by comparing the anesthetized, bled group (AB) to the conscious, bled group (CB).

Statistical significance was accepted at p<0.05. The data is expressed as mean \pm SEM.

171- RESULTS

Six pigs died before the experiment: 2 from aortic ruptures because of the flown ster, and 4 from cardiac fibrillation during surgery which occurred at the time of suturing the strain gauge to the left ventricle in spite of prophylactic xylocaine treatment. We were thus compelled to omit the strain gauge technique. Two pigs were excluded because they developed a perforated ulcer with uncontrolled blood loss.

We originally planned to remove 24 ml/kg blood volume, but all the anesthetized pigs (three) died. So we used a smaller hemorrhage

study all the pigs throughout the recovery period, we decided to perform a 15 ml/Kg hemorrhage.

Controls values :

Control levels were similar to those reported in previous published studies, except parameters wich were changed because of the operations (enzymology). Depending on whether comparisons were made on the acute or recovery phases of the study, two control values were used. For the acute (0-360 min) phase, the first point before the experiment but after anesthesia administration was used. For the recovery (24 to 96 hours) phase, the point before anesthetic administration was used. To allow comparisons, the control data point for each variable was assigned a normalized value of 0 and all other values were corrected to reflect relative changes. All the values (mean and SEM) are submitted in the appendix. The interesting changes are shown in curve form (figures 1 to 5).

Memodynamics responses :

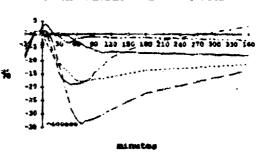
Baseline values of all physical parameters did not change during the time-control, pre-injary and/or pre-hemorrhage periods. Figure 1 shows the changes in heart rate (HR), mean aortic pressure (MAP), central venous pressure (CVP) and cardiac output (CO). Hemorrhage caused a significant (p<0.05) 20% increase in HR between initial and post-hemorrhage values only in the anesthetized, non-injured bled group (AB). There were no time-related changes in HR in any of the other four groups (A, AI, AIB and CB). For the between group comparisons, the AB group had a significantly higher HR when compared to the control group (A vs AB, p<0.05) as well as the conscious bled group (AB vs CB, p<0.01). Ballistic injury had no effect on HR, either by itself or in combination with hemorrhags.

This level of hemorrhage caused MAP to fall significantly, both with respect to time and when compared to the time control group, by 20% in the conscious group (p<0.05) and by 30% in the anesthetized group (p<0.01), and MAP did not return to control levels even after 240 min. There was a heterogenous response (initial decrease and then

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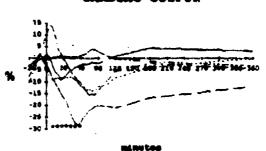
MEAN ARTERIAL PRESSURF



CENTRAL VENOUS PRESSURE



CARDIAC OUTPUT



Hemodynamic changes in the five groups: Anesthetized (----), Anesthetized and injuried (----), anesthetized and bled (----), enschetized, injuried and bled (----), conscious bled (----) (time of hemomrhage: ******)

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which did not permit us to detect a significant difference. As with HR, ballistic injury had no effect on MAP, either by itself (A vs AI) or in combination with hemorrhage (A vs AIB). This suggests that ballistic injury attenuates the fall in MAP induced by this level of hemorrhage in anesthetized animals.

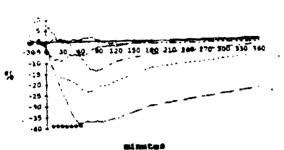
Central venous pressure (CVF) decreased significantly (p<0.05) between pre and post-treatment values in all treated groups: by 45% in the AB group, 40% in AIB, 35% in CB, and 25% in the AB group (0.05 < p < 0.10). The CVP recovered toward control values at 2 h after hemorrhage in the answthetized bled group (AB) but not in the injury alone group (AI) or the combination group (AIB). Between-group comparisons showed significant differences between A vs AI (p<0.01), A vs AIB (p<0.05), and AB vs CB (p<0.05).

Cardiac output (CO) was significantly (p<0.05) reduced from prehemorrhage values by 25% in the anesthetized bled group (AE). Although there was a tendency for CO to fall in the order groups (by 12% in the combination AIB group and by 10% in the conscious bled (CB) group), the responses were heterogenous and did not permit us to find statistical significance. The anesthetized hemorrhage group (A vs AB) had a significantly (p<0.01) greater response than the combination of ballistic injury and hemorrhage group (A vs AIB), again indicating that the ballistic injury attenuated the fall in CO in response to the hemorrhage. There was also a significant (p<0.01) difference between the anesthetized and conscious bled groups (AR vs CB).

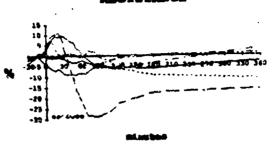
Figure 2 shows the changes in stroke volume (SV), systemic vascular resistance (SVR), renal blood flow (RBF) and renal vascular resistance (RVR).

Stroke volume (SV) was significantly (p<0.05) reduced between pre and post-hemorrhage values in both the anesthetized (35%, AB) and the conscious (20%, CB) hemorrhaged groups. SV remained lower than baseline for the entire 5 hour experimental period in the AB group.

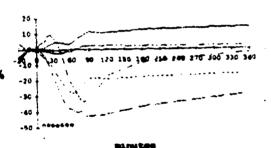
STROKE VOTUNCE



STSTEMIC VASCULAR RESISTANCE



REHAL BLOOD FLOW





Hernodynamic changes in the five groups: Anesthetized (----). Ancethetized and injuried (---), anesthetized and bled (----), anesthotized, injuried and bled (---), conscious bled (----)
(time of iremormage: ******)

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Hemorrhage reduced SV significantly (p<0.01) in the anasthetized bled group when compared to the time control group (A vs AB). Only the anesthetized bled (AB) group and the combination injury and hemorrhage (AIB) group decreased SVR significantly (p<0.05) by 25t and 10%, respectively, between initial and post-hemorrhage values. There were no significant differences between any of the groups at the end of hemorrhage (AI vs AIB, AB vs AIB, AB vs CB).

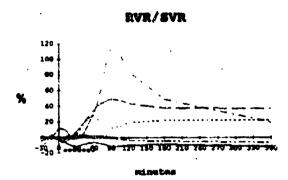
There was a significant (p<0.05) decrease in renal blood flow (RBF) by 40% in the anesthetized bled group (AB) and by 30% in the conscious bled group (CB) between the initial and post-hemorrhage values. Hemorrhage group had a significant (p<0.01) decrease in RBF.

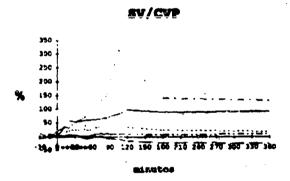
There was no change in renal vascular resistance (RVR) in any group between control and post-hemorrhage values. The increase in RVR in the combination (AIB) group was caused by heterogenous values during the period (after 60min) when the animals were recovering from anosthesia. There were also no significant differences between groups.

Figure 3 shows the ratios of RVR/SVR and SV/CVP.

There was a significant (p<0.05) increase in the RVR/SVR ratio of 35 in the AB group between the control and post-hemorrhage values. The RVR/SVR ratio was significantly (p<0.05) greater in the A vs AB group but not in the A vs AIB group. The RVR/SVR ratio was also significantly greater in the AB group compared to the CB group. There were no other significant differences between any of the other groups.

There was a significant (p<0.05) increase in the SV/CVP ratio, by 60% in the AI group and by 100% in the AIB group between the pre and post-hemorrhage values. The increase by 320% in the AIB group was caused by a heterogenous response during the period when the animals were recovering from anesthesia. Ballistic injury increased significantly ratio SV/CVP, either by itself (p<0.01) or in combinaison with hemorrhage (p<0.05). There were no other significant differences between any other groups.





Hemodynamic changes in the five groups: Ansithetized (---), Anesthetized and injuried (---), anesthetized and bled (---), anesthetized injuried and bled (----), conscious bled (----) (time of hemomage: *****)

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Only the parameters with significant variations are shown in the figures 4 and 5.

Hemorrhage produced an increased release of epinephrine with significant (p<0.05) differences at +60 min in all three hemorrhage groups (AB, CB, and AIB) when compared to the pre-hemorrhage value. The comparisons between the AI vs AIB, the A vs AB, and AB vs CB groups were significant (p<0.05).

Glucose was increased by 35%, 25% and 10%, respectively in the three groups: AB (p<0.01), AIB (p<0.01), and CB (p<0.05) groups when compared to pre-hemorrhage values. There was a late 20% increase in glucose in the anesthetized and trauma alone (AI) group at 360 min (p<0.01). There were no significant differences between groups. Plasma creatinine increased (p<0.01) by 38% in AIB group and by 18% in AB group at 180 min. At this time, there were significant (p<0.01) differences between the AI vs AIB, A vs AB, AB vs CB, and A vs AIB groups.

Blood PH and blood gases didn't shown significant variations between pre and post-hemorrhage values. However, plasma bicarbonate was decreased significantly (p<0.05 at + 180 min) in both the AB and AIB groups. Six hours after hemorrhage, arterial bicarbonate tended to return toward its control value but only in the AB group. At this time, arterial bicarbonate was decreasing in the AI group. There were no significant differences between groups.

Figure 5 shows some biological parameters which presented some significant long-term changes, to 96 hours after the experiment.

Hematocrit and hemoglobin levels fell rapidly until 6 hours (p<0.01) and more moderatly until 24 hours in the 3 bled groups (AB, CB, and AIB). At this time, there were no statistically significant differences between those groups.

The number of leukocytes presented a moderate increase (10 to 25%) at 6 hours, and a second larger increase (+45 %) at 72 hours in the AIB group. These changes were significant. There were no significant changes between the groups.





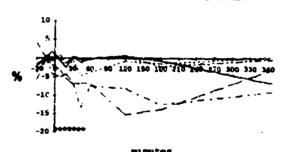


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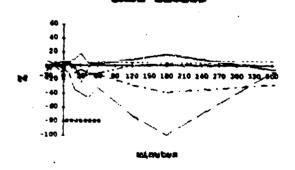
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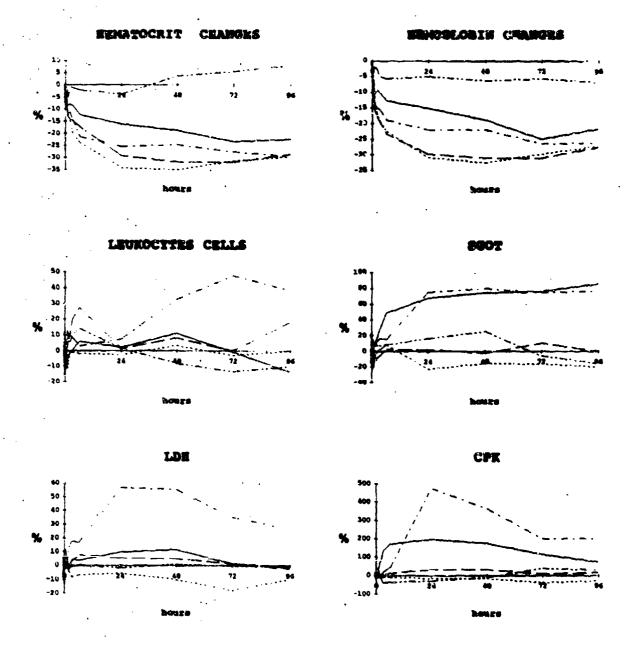


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Biological changes in the five groups: Anesthetized (---), Anesthetized and injuried (---), anesthetized and bled (---), anesthetized, injuried and bled (---), conscious bled (---) (time of hemomage: ******)

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Biological changes in the five groups: Anesthetized (---),
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(time of hemorrhage: -----)

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Reflecting cellular destruction, the enzymes SGOT, LDH and CPK were increasing to a peak at 24 hours in both trauma groups (AI and AIB). The increase in the AI group was similar to the AIB group for SGOT, put somewhat less for LDH and CPK.

IV- DISCUSSION

For this study, we used the splecnectomized pig model. This model is considered as one of the best with respect to hypovolemia, and also the best with respect to studying projectile injury (20). In fact, the muscular thickness of the thigh (about 14cm), allows one to obtain a significant cavitation effect (4) and therefore the resulting tissue lesions are more representative of those which are observed in humans (1). Furthermore, we have chosen the site of impact so that there will neither be a diaphyseal fracture (distance between diaphysis and projectile trajectory > 7cm), nor damage to a major blood vessel. Under these conditions, the hemorrhage due to the damage from the projectile is minimal. The ballistic injury is administered just before the hemorrhage, thus making these conditions more realistic to those seen in combat. In creating a ballistic injury, the rules of good laboratory practice require the experimenter to anesthetize the animals. We have chosen to use Ketamine as the anesthetic be:ause it modifies the function of the cardiovascular system minimally during hypovolemia (25, 26, 27). After administration of the anesthetic, we have found a slight increase in heart rate, mean arterial pressure, and aortic flow. These results, in agreement with those given in references, are consequence of central stimulation of the sympathetic system (24, 26, 25). In preliminary measurements of the plasma volume using the Evan's Blue method, we showed that the total blood volume of the splenectomized, 90 kg, Large White Pig, varies between 60 to 66ml/kg. A hemorrhage of 15 ml/kg therefore corresponds to a moderate hemorrhage of 25% of the total blood volume. Such a blood loss does not provoke a maximal sympathetic stimulation; this allows us to compare the amount of stimulation evoked by different treatments.

In the conscious animal, a hemorrhage of 25% provokes a very mild response. Because of an insufficient number of animals in this study, the changes observed in the conscious animals were frequently statistically insignificant. Likewise, in the anesthetized animals, we have found some physiological responses of variable intensities. Nevertheless, in either conscious or anasthetized animals, the physiological responses to the hemorrhage were generally in accord with those given in the literature (10, 13, 2, 16). The response corresponds to the classic scheme of a moderate sympathetic reaction: the reduction of blood volume and arterial pressure stimulates the sympathetic system via the volume and baroreceptors, which immediately causes an increase in heart rate and vasoconstriction of certain peripheral vascular beds like the kidneys. As found in many other animal studies, the decrease in renal flow was independent of the decrease in aortic flow. The variable, RVR/SVR, gives a good it a of this differential vasoconstriction. In contrast, we observed that the systemic vascular resistance decreased in this study. Hannon has reported similar results in the literature of porcine hypovolemia. The variability of the physiological parameters found in the conscious and anesthetized hemorrhaged groups generally agree in some ways but are of different intensity: they are more marked when the animal is anesthetized. In fact, the administration of anesthetic, general, is a factor which aggravates the response to hypovolemia. There exist many hypotheses concerning the mechanisms of action of ketamine: it can dimirish the vasometer responses, in spite of its effacts on stimulating sympathetic discharge centrally, and it has a depressant effect on the myocarumum (24). Our study shows effectivel, a large drop in the stroke volume (the increase in heart rate wasn't sufficient to be the cause) in animals anesthetized with ketamine.

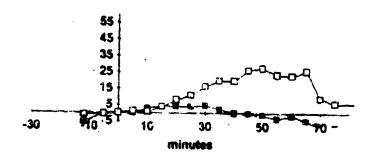
The projectile used in this study has a speed between 900 and 950m/sec at impact. The injury caused to the soft tissue of the thigh shows those local hemodynamic modifications as described by Rybeck, Holmström and Almskog: a large zone of hyperemia surrounds the ischemic area adjacent to the projectile trajectory. Thus, in the animals of the AI and AIB groups, we saw a cutaneous vasomotor reaction (RVMC of Breteau) appearing some minutes after the injury,

giving evidence of the existence of this phenomenon in the present study. Rybeck has shown that the local lesions are accompanied by an increase in the loss of blood in each of 2 limbs, with a slight reduction of arterial pressure and heart rate. It suggests the possible action of vasodilatory or neurogenic substances, but does not explain the non-cardiac response.

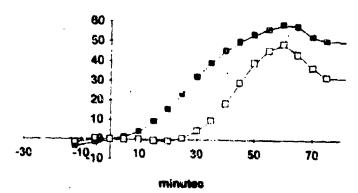
In the anesthetized and ballistically wounded animals, we have found a consistent decrease of the CVP, the other parameters do not present significant variations. The CVP decreased rapidly until 30min after the injury and it was maintained at this low level. This drop indicates an increase in the venous capacitance which could be due to the slow liberation of vasoactive substances by the injured tissue.

The animals in the injury and hemorrhage group presented rather heterogenous responses, which explains their occasional lack of statistical significance and makes it difficult to interpret. Nevertheless, all show a drop in CVP like those observed in the anesthetized, injured without hemorrhage. One would therefore hypothesize, as we have noted with the 18 ml/kg hemorrhage experiment, that the injury would aggravate the effects of hemorrhage. But this study has shown that for the hemorrhage of 15ml/kg, the arterial pressure subsides less \mathbf{v} v there is trauma associated with it. Two hypotheses can be advanced : either (1) periphetal vasoconstriction, or (2) the inotropy of the heart appears more effective in this case. Evidence against the first hypothesis, only 1 of 5 animals reacted with an intense vasoconstriction (the reason for the large response observed in the AIB group on the RVR and RVR/SVR curves. The other subjects appear to have maintained their arterial pressure with an increase in inotropy. Thus, in spite of a low preload, the stroke volume dis not drop significantly, contrary to that observed in the anesthetized hemorrhaged group. The results which we have obtained of the animals instrumented with the myocardial strain gauge appears to confirm this hypothesis. Figure N° 6 shows the time course of the change in heart rate on the bottom panel and the relative change in the ratio of cardiac strain gauge over stroke volume on the top panel, for one animal in the AB group and one animal from the AIB group. This figure gives evidence for the inotropic hypothesis: one sees that

RATIO STRAIN GRUGE/SV CRAUSES (%)



MEANT RATE CHANGES (8)



Comparison of the strain gauge/stroke volume (reflection of myocardial inotropy) and the heart rate changes in the same animal from 2 groups: anesthetized with hemorrhage (-a) and anesthetized, wounded with hamorrhage (-b).

The service databased ratio increased when there was an injury,

with the injury confirming the previous hypothesis.

the heart rate increased later. It the heart rate increased later to the heart responds almost exclusively with an early increase in the heart rate. The ratio SV/CVP has been calculated to evaluate the increased significantly

It appears therefore, that in the case of a hemorrhage preceded by a ballistic injury, the heart used the mechanism of its increased inctropy, rather than its rate, to compansate for the hypevolemia. This reaction, which is effective for a hemorrhage of low volume, can become unsufficient or result in cardiac fatigue for a larger hemorrhage, as we have observed in the animals injured and hemrrhaged at 19ml/kg, which died at the end of hemorrhage. However, to confirm this hypothesis, additional experiments will be necessary.

Inotropy and heart rate are modulated by the vagal and sympathetic activity. A modification of the responses of these parameters can come from the modulation of the balance between the two system's central control nuclei. Also, it will be particularly interesting to study the precise role of the vagus, by vagotomy, injection of atropine, or a dote of pancreatic polypeptide.

The modifications of hematologic and biochemical parameters reflect the physiological responses which resulted from the injury and/or the hemorrhage: the drop in hematocrit and the rate of hemoglobin are evidence of transcapillary refill. The epinephrine concentration reached its maximum at the end of hemorrhage reflecting increased sympathetic activity. The increase of the creatinine is a consequence of renal disfunction. The blood gases and pH, the hydroelectric equilibrium did not vary in a significant fashion. In contrast, the bicarbonate and the base excess dropped moderately, which corresponds to a well compensated hypovolemia. With the animals injured and hemorrhaged, the enzymes, evidence of cellular lysis, are liberated in a slower fashion but more intensely than in the wounded but not hemorrhaged group, reflecting the differences of local perfusion.

In conclusion, an injury of soft tissue by a projectile under anesthesia, modifies the physiological responses of the organism to

with hemorrhage, the heart utilizes the mechanism of increased increased increased the hypovolemia. This compensatory reaction, effective for the small loss of blood (25%) became insufficient with a larger hemorrhage. The model of simple hemorrhage is therefore not quite representative of that which one would observe on the battlefield where hemorrhage is frequently the consequence of a bullet wound. There may be some limit in extrapolating the conclusion gained from this anesthetized, combintion wound-hemorrhage model to that expected in the conscious animal, because the anesthesia itself modifies appreciably the responses to hemorrhage.

ACKNOWLEDGENENT,

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